# ECHOCARDIOGRAPHIC EVALUATION OF MITRAL E/e' AS A PROGNOSTIC INDICATOR IN ST-ELEVATION MYOCARDIAL INFARCTION

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*In partial fulfilment of the requirements for the award of the degree of*

**D.M. CARDIOLOGY**

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**THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI, INDIA**

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# CERTIFICATE

This is to certify that the dissertation titled **ECHOCARDIOGRAPHIC EVALUATION OF MITRAL E/e' AS A PROGNOSTIC INDICATOR IN ST-ELEVATION MYOCARDIAL INFARCTION** is the bonafide original work of Dr**. R.RAMESH**,in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2014.The period of post-graduate study and training was from August 2011 to July 2014.

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# DECLARATION

I, **Dr.R.RAMESH**, solemnly declare that this dissertation entitled, "**ECHOCARDIOGRAPHIC EVALUATION OF MITRAL E/e' AS A PROGNOSTIC INDICATOR IN ST-ELEVATION MYOCARDIAL INFARCTION**" is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2011 – 2014 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor M.S.Ravi, M.D.D.M. This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology**.

Place: **SIGNATURE OF THE CANDIDATE**

Date:

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# CONTENTS

## ABBREVIATIONS AND ACRONYMS

STEMI –ST-elevation Myocardial Infarction

AMI - Acute Myocardial Infarction

TIMI - Thrombolysis In Myocardial Infarction

LVEF - Left Ventricular Ejection Fraction

MV - Mitral Valve

E - MV early diastolic velocity

A - MV late diastolic velocity

e ‘ - Tissue Doppler early diastolic velocity of lateral mitral annulus

DT - Deceleration Time of early diastolic mitral inflow.

TDI -Tissue Doppler Imaging

PCWP - Pulmonary Capillary Wedge Pressure

mLVDP – mean LV diastolic Pressure

LVEDP - LV End Diastolic Pressure

# INTRODUCTION

STEMI is a major public health problem in the developed world and is rising in developing countries . In the United States, almost 1 million patients per year suffer from an acute MI, and more than 1 million patients with suspected acute MI yearly enter coronary care units in the United States. The rate of MI rises for both men and women sharply with increasing age, and racial differences exist, with MI occurring more frequently in Asian men and women, regardless of age . Of particular concern from a global perspective, the burden of MI in developing countries may approach those now afflicting developed countries. Limitations in available resources to treat STEMI in developing countries requires major efforts to strengthen primary prevention programs

## Improvements in Outcome

Mortality from STEMI has declined steadily. This drop in mortality appears to result from a fall in the incidence of STEMI and a fall in the case fatality rate once STEMI has occurred.

Several phases in the management of patients have contributed to the decline in mortality from STEMI. The “clinical observation phase” of coronary care emerged in the first half of the 20th century and focused on a  recording of physical and laboratory findings,. The “coronary care unit phase” began in the mid-1960s and was notable for detailed analysis and management of cardiac arrhythmias. The “high-technology phase” heralded by the introduction of the invasive balloon catheters set the stage for bedside hemodynamic monitoring and more precise hemodynamic management. The modern “reperfusion era” of coronary care was introduced by intracoronary and then intravenous fibrinolysis, increased use of aspirin, and the development of primary percutaneous coronary intervention

## RISK ASSESSMENT AFTER ACUTE MYOCARDIAL INFARCTION

Patients with ST-elevation myocardial infarction (STEMI) should undergo risk stratification soon after presentation. Since most patients with STEMI undergo reperfusion therapy, early risk stratification and late risk stratification attempts to identify patients who are at increased risk of death. Risk stratification is accomplished with the use of validated risk prediction models that include the most important predictors of outcome.

**EARLY RISK STRATIFICATION** — All patients with ST-elevation myocardial infarction (STEMI) should undergo risk assessment within the first four to six hours of hospitalization. However, even low-risk patients should undergo primary reperfusion ( with percutaneous coronary intervention or thrombolysis) in a timely manner.

The TIMI risk score and the GRACE risk model are two commonly used scoring systems used in patients with STEMIin patients admitted with an acute myocardial infarction (MI) are categorized into high risk groups on the basis of risk factors like age prior MI, Killip class, Anterior MI, Hypotension and Tachycardia.

**<u>Clinical utlity of Echocardiography in Acute Myocardial Infarction</u>.**

Assessment of resting left ventricular function is an important part of risk stratification in patients with acute myocardial infarction . Patients with left ventricular systolic dysfunction have increased mortality at six months and one year. The increase in mortality is most pronounced in the minority of patients with a ejection fraction (EF) ≤30 percent. In addition, patients with EF ≤35 percent are at increased risk for sudden cardiac death after MI and should be considered candidates for appropriate therapy. EF is usually measured before discharge. In patients with heart failure or suspected mechanical complication echocardiography may be indicated early in the hospitalization. However, measurements during hospital stay may be misleading, since improvement in EF, beginning within three days and upto 14 days, is common in patients who are reperfused. Two separate studies have shown that approximately 58 percent of patients significantly improve d EF after reperfusion in acute STEMI. This may reflect, recovery from myocardial stunning since it is associated with a reduction in the size of the myocardial perfusion defect. Patients with improved EF may have significantly lower mortality than those who show no improvement (1.2 versus 5.6 percent at three years)

In the setting of acute myocardial echocardiogram is a favoured diagnostic tool to identify poor prognostic factors like right ventricular infarctions, mitral regurgitation, left atrial enlargement and diastolic dysfunction.

Assessment of diastolic dysfunction with the help of echocardiography is used in the diagnosis of diastolic heart failure (HF) and elevated filling pressures in the LV which is an indirect measure of the Pulmonary Capillary Pressure.

## AIMS & OBJECTIVES

1. To analyze the clinical characters, in hospital events in patients admitted with acute wall myocardial infarction (STEMI) evaluated by echocardiographic color and tissue doppler E / e’ ratio evaluation of mitral valve as a measure of left ventricular filling pressure.

2. To assess the usefulness of E/e’ as a prognostic indicator in patients STEMI with or without revascularisation therapy to predict hospital mortality.

## REVIEW OF LITERATURE

Variable degrees of systolic dysfunction is prevalent after a myocardial injury and the pathophysiological principles s underlying this dysfunction and its effect on the outcomes of the patient has been the subject of several path breaking studies which have contributed to modification in therapeutic strategies in a radical manner.

Moreover, heart failure demonstrated by clinical or radiographic evaluations over and above decreased systolic function is strong predictor of prognosis after myocardial infarction. Seemingly beningn myocardial damage can lead on to signs of pulmonary congestion indicating elevated ventricular end diastolic pressure.

This has been attributed to impaired active relaxation of the myocardium and increased chamber stiffness leading onto abnormal diastolic function.

This has to be determined by invasive cardiac catheterisation studied with the use of micromanometer catheters such an invasive strategy is not practical for use on a day to day basis.

Similarly directly measuring the right heart or left heart end diastolic pressures is useful in predicting  worser outcomes after myocardial infarction. Their routine usage is not recommended due to high incidence of complications.

In order to circumvent these issues, the use of non-invasive methods in the assessment of diastolic function like Doppler – echo cardiography as a surrogate marker of abnormal ventricular filling has come up on a large scale. These observations can be used to predict prognosis after myocardial infarction in such patient groups.

The following brief discussion about the current understanding of ventricular filling after infarction reviews the usage of left atrial volume and filling, mitral Doppler dynamics and its abnormal patterns in the setting of an acute myocardial injury using two dimensional and Doppler echo cardiography.

**Doppler Echocardiography**

Doppler technique helps in evaluation of flow velocities  by precisely locating specific points inside the various regions of the heart and grading the flow.

Velocity of blood flow across the mitral valve is determined by the ventricular negative pressure generated and the difference in pressure between the left atrium and ventricle.

This initial phase is followed by a progressive decline in the velocity of inflowing blood. This phase has a duration raging from 140-240 milliseconds and is called the mitral deceleration time (DT) (Figure 2). After a brief period of diastasis, atrial systolic produces an rise in velocity of the inflow which is much less than that compared to the early inflow.

As a result, the ratio between the early and late inflow velocities across the mitral valve (E/A ratio) is in the range of 1 to 1.5.

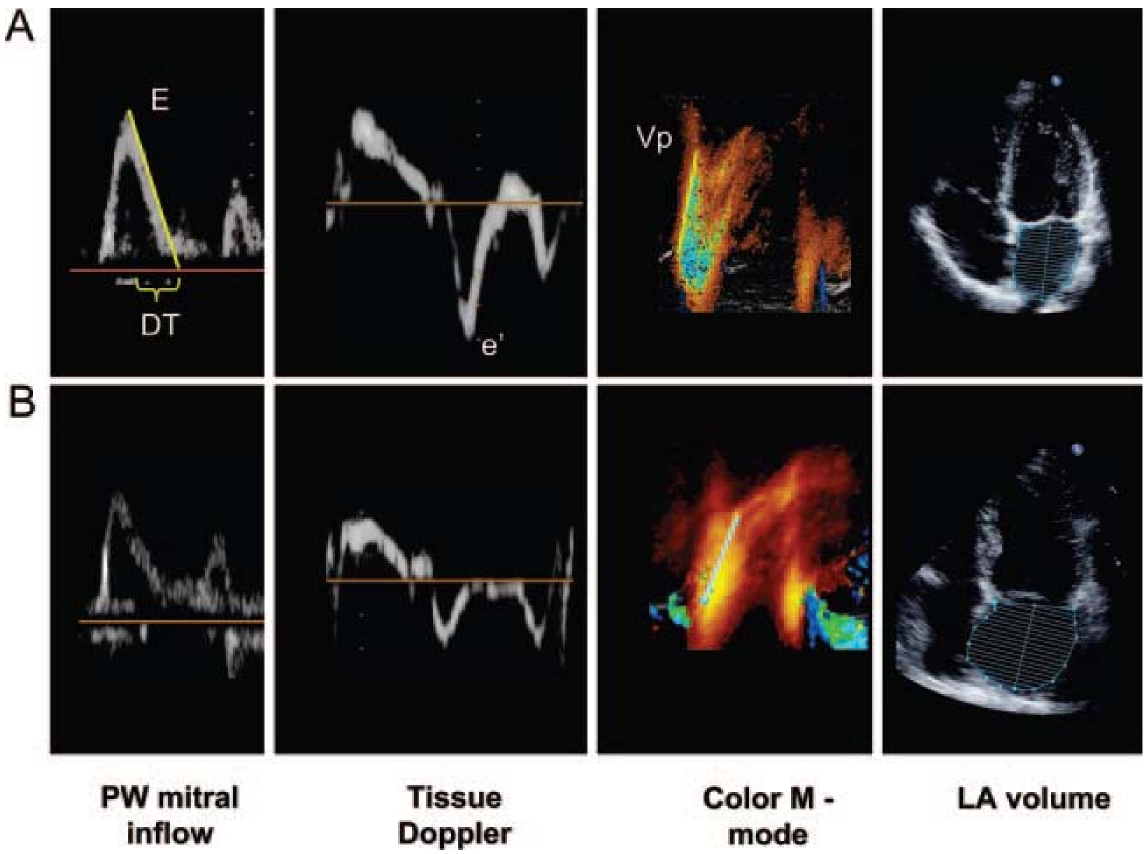
The early mitral inflow might decrease as a result of impaired active relaxation leading onto augmented atrial contribution to filling of ventricules. This causes reversal in the E/A ratio with concomitant prolongation of DT (Figure 2). As the diastolic function progressively worsens the left atrial pressure begins to rise creating increasing gradient between the left atrium and the ventricle. This causes rise in velocity of early inflow even though the active relaxation is impaired. As a result of rapid equilibration of pressure, early part of the ventricular filling comes to a close prematurely with a decrease in the time period of early diastolic filling returning the DT to normal intervals. This seemingly normal mitral inflow pattern even in the presence of impaired relaxation and increased left atrial pressure is called the pseudonormal pattern (Grade 2 dysfunction) (Figure 1).

With further worsening of active relaxation, early diastolic filling abruptly terminates because of worsening left ventricular stiffness.

The deceleration time will be fore shortened and as a result the E/A ratio is increased. This pattern is termed restrictive filling (Grade 3 dysfunction) (Figure 2). This can assume two forms as reversible and irreversible on the basis of response to reduction in preload by treatment or by valsalva maneuver. If these interventions cause reversal of the pattern of the filing is called reversible and irreversible if these interventions produced on effect.

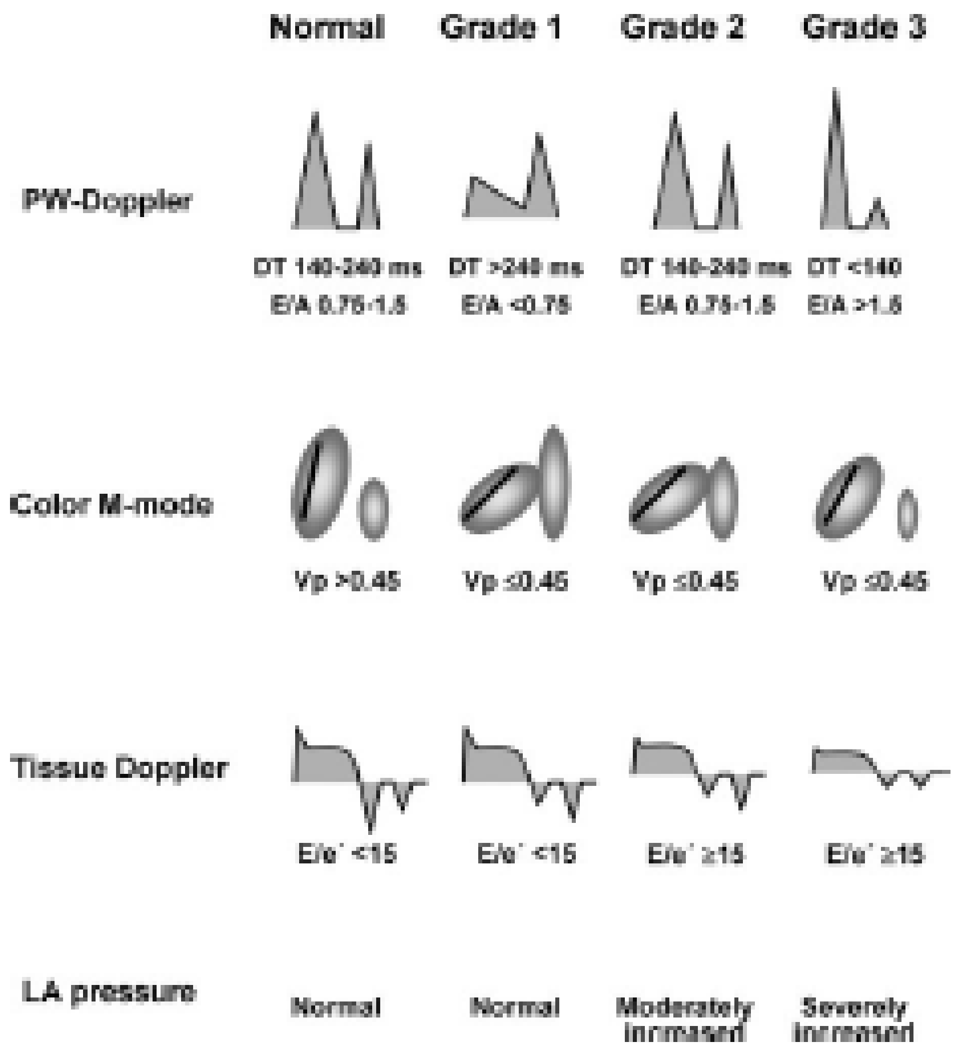
Although mitral Doppler inflow patterns continue to be the bedrock on which ventricular diastolic function can be assessed, there are certain short comings to this measurement. They can rapidly change with variations in blood volumes. A pseudonormal pattern even in the event of an elevated diastolic pressure is another disadvantage. To circumvent these, parameters that are load-independent can be used like, the Doppler inflow patterns in the pulmonary veins. This is again hampered by irregular heart rates. Other parameters like propagation of blood flow into the left ventricle studied the help of color M-mode. Lastly tissue Doppler evaluation of movement of the mitral annulus in the diastolic phase is a promising new modality to assess diastolic function.

FIGURE .1 ECHO ASSESSMENT OF DIASTOLIC FUNCTION




1. Mitral inflow Doppler depicting E and A waves and DT.
2. Tissue Doppler of mitral annulus showing e’ velocity.
3. Color M-mode showingVp velocity.
4. 2-D Echo showing LA volume.

FIGURE 2. GRADES OF DIASTOLIC DYSFUNCTION



## Tissue  Doppler Echocardiography.

Myocardial tissue motion can be studied with the aid of tissue Doppler imaging which demonstrates the tissue motion parallel to the beam of Doppler waves.

The ventricular apex is fixed during cardiac cycle resulting in a movement of the basal regions of the ventricle in a parallel direction with the long axis of the heart. The ventricular motion along the basal region of the heart is an indicator of longitudinal axial contraction.

The early diastolic mitral motion expressed as e’ velocity is a predictable indicator of ventricular relaxation. Catherization studies show that e’ has an inverse relationship with the measures of ventricular relaxations. These velocities are less affected in the presence of changes in ventricular volumes and can help in the assessment of pseudonormal ventricular fillings.

The routine use of ratio between E velocity and early diastolic mitral tissue velocity (E/e’) in the assessment of ventricular end-diastolic  pressures has been extensively studied in several reports.

Even in the presence of cofounding factors like atrial fibrillations, tachy cardia, normal and abnormal ventricular systolic function, ventricular hypertrophy, mitral regurgitation E/e' was good at predicting end-diastolic pressures. (Ommen et al) (9) showed that increased end-diastolic pressure is predicted with reasonable accuracy within an narrow range by the measurement of E/e'

The caveat is that tissue Doppler detects the velocity of an actively contracting myocardium and a passive tethering. Among the two sites used, the velocity of the lateral annulus is slightly higher than that of the septal counter part. Local myocardial injury might alter these regional velocities and hence wrongly predict myocardial motion and ventricular functions.

Diastolic dysfunction is not infrequent in acute myocardial infarction and substantially contributes to evaluation of prognosis. Doppler echocardiography is the most commonly used tool in the measurement of diastolic filling of the left ventricle. Examination of the mitral inflow can provide us with detailed indices for indirectly measuring ventricular diastolic pressures and assessing prognosis after myocardial infarctions.

But, the degree of ventricular relaxation, mitral valve characteristics, atrial compliance factors and pressure determine the mital inflow dynamics and hence confounds the interpretation of the mitral inflow parameters. As a result, the diastolic function is poorly assessed in certain subset of patients.

These short comings in using mitral inflow parameters can be overcome with the concomitant use of the Doppler indices like pulmonary venous Doppler inflow.

The mitral annular tissue Doppler evaluation is a wonderful tool to correctly assist these factors which confound flow velocities. This could predict diastolic filling pressures reliably in many large group of patients.

**Determination of ventricular diastolic pressures with the use of Doppler techniques.**

Measurement of ventricular diastolic pressures non invasively is a useful adjunct in clinical assessment of decrease in ventricular compliance as exhibited by shortening of DT and gradually rise in E/A ratio and elevated left atrial pressure.

Ventricular systolic function also alters mitral inflow patterns. With normal systolic functions, DT and E/A ratios reasonably predict ventricular diastolic pressure and fail to do so with abnormal systolic functions.

Mitral annular motion assessment using tissue Doppler is an emerging technique for evaluating cardiac function. The descent of the mitral annulus during cardiac contraction is recorded in two phases with pulse – wave Doppler.

These velocities are load independent and show an excellent relationship to both ventricular systolic and diastolic function.

E/e' is a useful measure of ventricular diastolic pressure with reasonably combines both ventricular relaxation and inflow velocities. However there is a grey zone in the value of E/e' between 8 to15.

Non uniform ventricular relaxation especially seen in patients with myocardial damage could confound the relationship between ventricular relaxation and measurement of annular velocity.

**Effect of ventricular Filling Pattern on Prognostic outcomes After MI**

The value of restrictive diastolic filling in acute myocardial infarction as a prognostic tool was originally studied by Oh et al (10). In his study of a group of patients, a higher prevalence of heart failure was seen in patients with restrictive filling patterns.

Poulsen et al (11) in his study again confirmed these findings and observations in patients admitted with acute myocardial infarction.

Nijland et al (12) similarly showed that patients with a DT of less than 140 milli seconds had a reduced survival compared with patients with non restrictive filling patterns.

Restrictive filling defect was more prevalent in patients with older age, severe systolic dysfunction, and these groups are more prone for heart failure during the hospital stay.

Irrespective of whether patients had (NSTEMI), non – ST elevation MI or ST elevation MI, whether they underwent thrombolytic therapy or angioplasty, restrictive filling, reliably predicted prognosis.

Clinical variables like heart failure, ventricular systolic function did not alter the utility of using DT as a prognostic tool. Two other studies (17,18) have shown that a shortened DT independently predicted worser outcomes after acute myocardinal infarctions over and above those routinely used prognostic factors like age, sex, Killip Class, ejection fraction, infarct size or wall motion scores.

A DT less than 140ms or E/A less than 2 was considered restrictive filling and its influence on outcomes after myocardial infarctions has been studied in detail (22).

Mild diastolic dysfunction is emerging as an independent variable associated with worse outcomes after myocardial infarction. Evaluation of diastolic pressures is more important than mere presence of diastolic dysfunction which nails the relationship between diastolic function and prognosis after acute myocardial infarction (19).

**<u>Effect of elevated and diastolic pressures on the prognosis after myocardial infarction</u>**

Since direct measurements of diastolic ventricular pressures are difficult to obtain, non-invasive evaluation of ventricular diastolic pressures using E/Vp or E/e' could come in handy. This was recently shown in a retrospective study of a group of patients with myocardial infarction where an elevated E/e' ratio accurately predicted mortality in addition to EF, age, and a restrictive filling [19] .More importantly, E/e' was used for risk stratification in patients groups with normal as well as abnormal ventricular systolic function. Patients with pseudonormal filling (moderate increase in filling pressures)despite preserved LV systolic function have worser outcomes and this finding concurs with the results of studies in which the E/Vp ratio was used[.20,21]

## Left ventricular diastolic pressure and its prognostic value in acute myocardial dysfunction

Elevated ventricular diastolic pressures is associated with a high incidence of death after acute MI (23–25). Higher ventricular diastolic pressures suggest larger area of myocardial damage with more severe systolic dysfunction (26-29). Morover, ventricular pressure overload predisposes to ventricular, which would portend an worser outcome.

Apart from its prognostic value, the evaluation of ventricular diastolic pressures requires invasive measurements. On the other hand, Doppler echocardiographic assessment of mitral flow provides us with a non-invasive method of pin pointing patients with elevated left atrial pressures . Mild degrees of diastolic dysfunction is shown by impaired relaxation (without elevation of ventricular diastolic pressures). This suggests that the ventricle takes longer to fill (lengthening of DT), augmented by the atrial component of diastolic filling (reducing the E/A ratio). As diastolic function worsens further it is associated with rising left atrial pressures. Leads to a higher diastolic gradient across the mitral valve, resulting in abrupt equalization of the pressures in the left atrium and ventricle.

Advanced diastolic dysfunction is a poor prognostic indicator portending with a worser outcome after acute MI, exemplified abbreviated DT which is very specific. They support the well-established prognostic value of clinical indicators of ventricular diastolic pressures, such as Killip class.

**Value of E/e' as a prognostic indicator .**

The study by Hillis et al shows that, in the setting of acute MI, elevated E/e' reasonably correlated with conventional transmitral Doppler evidence of elevated ventricular diastolic pressure. More over it is a better prognostic indicator. This is in consonance with studies that demonstrated that E/e' better correlated with invasive measurement of left ventricular end diastolic pressure.(19). E/e' was also better correlated with Killip class on admission but, emerged to be a better predictor of survival. Again, this is because as there exists only a limited correlation between clinical features and invasive hemodynamic measurements of ventricular pressures, with the latter having prognostic superiority . Mitral diastolic flow velocities and DT correlate better with ventricular diastolic pressures in patients with impaired LV systolic function. But in patients with preserved LV systolic function, its value is much less. In contrast, the E/e' ratio correlates well with diastolic pressure, even in patients with a normal LVEF (19). An E/e'ratio >15 was the most useful predictor of an worse outcome, regardless of EF , the presence or absence of ST-segment elevation, or drug treatment on hospital discharge.

The E/e' ratio was superior to all known measures of LV systolic function, such as EF and Wall motion scores, for prediction of prognosis. However, it is prudent to understand that measurement of E/e'provides only additional prognostic data, with the greatest knowledge obtained by combining this information with clinical, systolic, and conventional diastolic parameters.

## MATERIALS AND METHODS

**Study centre :**

Coronary care unit of the department of cardiology , Government General Hospital, Chennai.

**Study design :**

Prospective ,observational and cross-sectional study

**Study sample:**

Consecutive patients admitted in coronary care unit between Jan 2014 and March 2014 at the department of Cardiology, RGGGH, Chennai with a diagnosis of ,STEMI ( ST-Elevation Myocardial Infarction ). [N = 50]

**Study Methods :**

Detailed history and cardiovascular examination was done with informed consent from the patient.

Patients were followed- up from admission to hospital discharge.

Echocardiography with color Doppler and tisuedoppler imaging of mitral valve will be done. The following echocardiographic parameters were studied in detail within 24 hours of admission by a single operator.

1.EF

2.LV Dimensions

3.Mitral E and A velocity (m/s)

4 Mitral e' velocity

5.Mitral E wave DT(Deceleration Time)

6.E/e' ratio.

**Stasticalanalysis :**

All the statistics were analysed using the SPSS version. 17 software and the tests used were Mean and SD, Chi-square test , Independent t-test and other relevant tools.

FIG .3 -Doppler E and A wave across mitral vavle

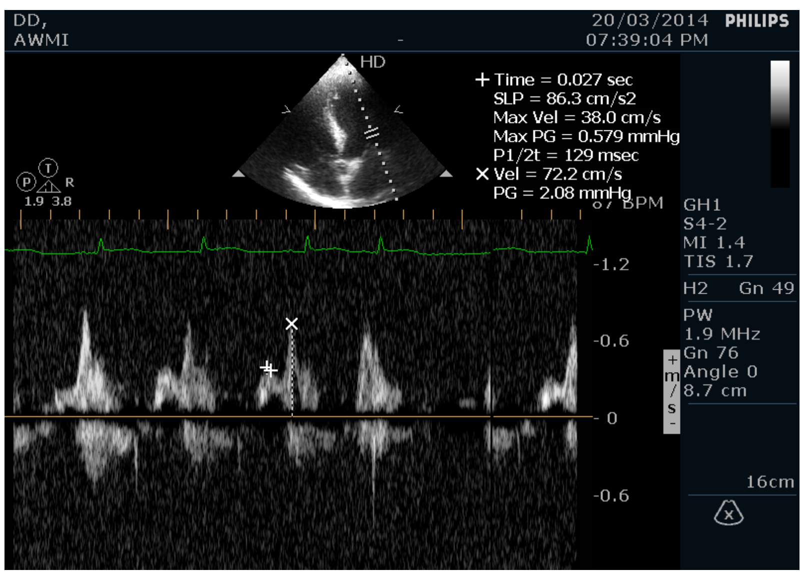


FIG. 4 -Mitral valve DT

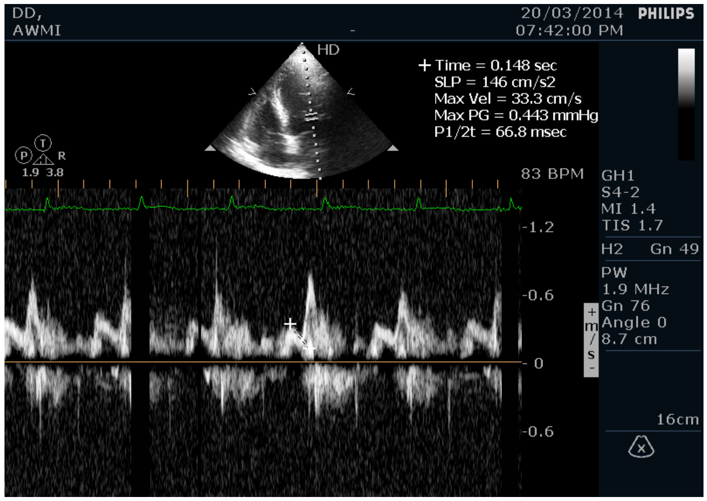



FIG .5 -Lateral annulus Tissue doppler

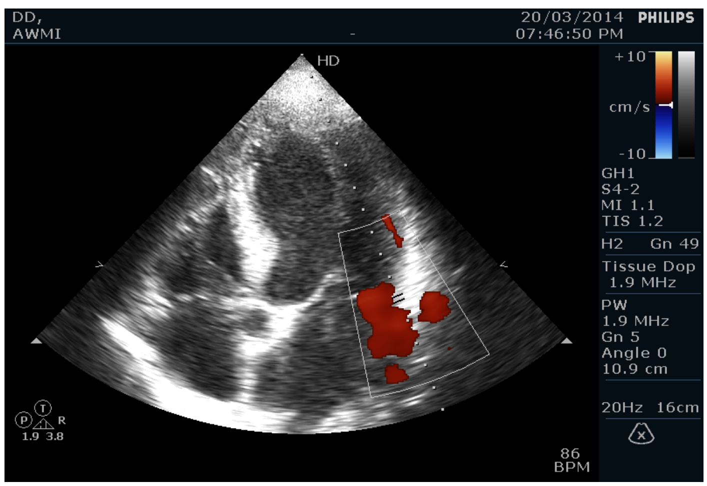
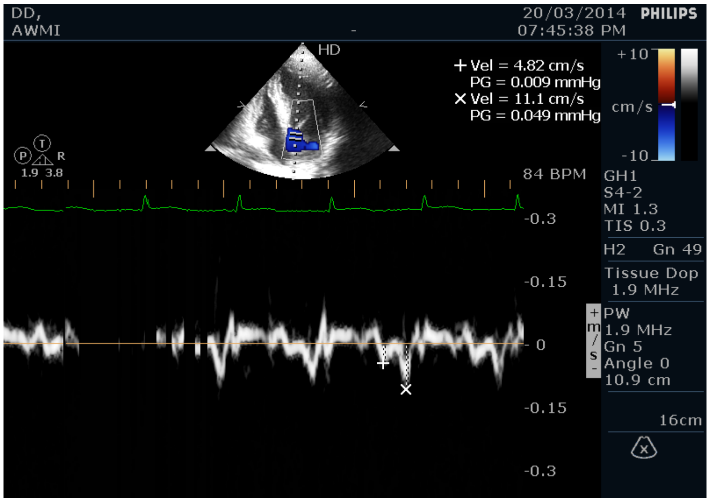


FIG.6 – Mitral e' and a' velocity



## ANALYSIS OF RESULTS AND DISCUSSION

Fifty consecutive patients admitted with the diagnosis of STEMI (ST-Elevation Myocardial Infarction ) were included in the study. Demographic ,clinical and echocardiographic findings of all these patients were analysed in detail and the following findings were observed.

80 % of the study population were males (n=40) and 20 % comprised of females (n=10).

TABLE.1 - SEX DISTRIBUTION

| | | N | % | Valid % | Cumulative % |
|---|---|---|---|---|---|
| Valid | Male | 40 | 80.0 | 80.0 | 80.0 |
| | Female | 10 | 20.0 | 20.0 | 100.0 |
| | Total | 50 | 100.0 | 100.0 | |

### AGE DISTRIBUTION OF PATIENTS

The mean age of the study populationwas 56.8 years in the overall group. While it was 54.9 years in the male population who entered the study it was higher in the female at 63.8 years .This was in conformation with all major studies involving cardiovascular events analysis.

| | | N | % | Valid % | Cumulative % |
|---|---|---|---|---|---|
| Valid | Below 40 | 4 | 8.0 | 8.0 | 8.0 |
| | 41-50 | 8 | 16.0 | 16.0 | 24.0 |
| | 51-60 | 19 | 38.0 | 38.0 | 62.0 |
| | 61-70 | 13 | 26.0 | 26.0 | 88.0 |
| | Above 70 | 6 | 12.0 | 12.0 | 100.0 |
| | Total | 50 | 100.0 | 100.0 | |

TABLE .2 AGE DISTRIBUTION OF PATIENTS

**RISK FACTOR ANALYSIS**

Among the commonly associated cardiovascular risk factors smoking was the most frequent associated risk factor accounting for 56 % of the patients involved in the study. Diabetes mellitus and hypertension accounted for 50 % of the patient group. Dyslipidemia was the least prevalent among the CV risk factors and constituted 44% of the study group.

<u>PREVALENCE OF CV RISK FACTORS</u>

TABLE.3 - SMOKING

| | | N | % | Valid % | Cumulative % |
|---|---|---|---|---|---|
| Valid | Yes | 28 | 56.0 | 56.0 | 56.0 |
| | No | 22 | 44.0 | 44.0 | 100.0 |
| | Total | 50 | 100.0 | 100.0 | |

TABLE .4 -DIABETES MELLITUS

| | | N | % | Valid% | Cumulative % |
|---|---|---|---|---|---|
| Valid | Yes | 25 | 50.0 | 50.0 | 50.0 |
| | No | 25 | 50.0 | 50.0 | 100.0 |
| | Total | 50 | 100.0 | 100.0 | |

TABLE .5 - HYPERTENSION

| | | N | % | Valid % | Cumulative % |
|---|---|---|---|---|---|
| Valid | Yes | 25 | 50.0 | 50.0 | 50.0 |
| | No | 25 | 50.0 | 50.0 | 100.0 |
| | Total | 50 | 100.0 | 100.0 | |

TABLE .6 - DYSLIPIDEMIA

| | | N | % | Valid % | Cumulative % |
|---|---|---|---|---|---|
| Valid | Yes | 25 | 50.0 | 50.0 | 50.0 |
| | No | 25 | 50.0 | 50.0 | 100.0 |
| | Total | 50 | 100.0 | 100.0 | |

**REPERFUSION STATUS**

Of the total of fifty patients studied, 41 patients received intravenous streptokinase as thrombolytic agent and pharmacological reperfusion strategy.

9 patients did not receive any reperfusion therapy either due to delayed presentation ( > 12 hours) or due to contraindications. One patient was subjected to a delayed invasive strategy because of a Ventricular Septal Rupture and persistent hypotension. His ventricular rupture was closed with a

percutaneous device ( AtrialSeptalOccluder ) and angioplasty and stenting to the culprit vessel was done.

TABLE.7- REPERFUSION STATUS

| THROMBOLYSED | NOT THROMBOLYSED |
|---|---|
| 41(n) | 9(n) |
| 82% | 18% |

## LOCATION OF MI

Majority of the patients studied in the group had an anterior wall STEMI than an inferior wall STEMI.60% of the patients had suffered an anterior wall myocardial infarction in comparison to about 40 % of the patients having suffered an inferior wall myocardial infarction.

TABLE .8 -LOCATION OF MI

| LOCATION OF MI | |
|---|---|
| **ANTERIOR** | **INFERIOR** |
| 30 (n) | 20 (n) |
| 60% | 40% |

Of the total of 7 deaths in the study population 4 patients had an inferior myocardial infarction and the remaining 3 patients had an anterior myocardial infarction. The mortality rates in their respective groups were 10 % and 25%. With the overall mortality at 14 % there was significantly higher mortality in patients who had suffered an inferior wall myocardial infarction than an anterior wall myocardial infarction.

TABLE .9 - DISTRIBUTION OF CHARACTERISTICS

| | N | Minimum | Maximum | Mean | SD |
|---|---|---|---|---|---|
| Age in years | 50 | 29 | 90 | 57.10 | 11.975 |
| TW (HRS) | 50 | 2 | 20 | 8.32 | 4.576 |
| LVEDD (mm) | 50 | 43 | 55 | 48.00 | 3.044 |
| LVESD | 50 | 28 | 46 | 35.24 | 4.926 |
| E (m/s) | 50 | .60 | 1.10 | .8108 | .10949 |
| A (m/s) | 50 | .55 | 1.10 | .7302 | .10570 |
| e' ( cm/s) | 50 | 4.2 | 9.6 | 6.794 | 1.6247 |
| E/e' | 50 | 7.5 | 20.0 | 12.620 | 3.7509 |
| E/A | 50 | .8 | 1.5 | 1.145 | .2001 |
| Valid N | 50 | | | | |

## **Distribution of Characteristics**

The age range of patients varied, with the youngest patient of 29 years of age and the oldest being 90 years, with a mean of 57.1 years

The median delay to hospital presentation also varied from 2 – 20 hours.

The various echo cardio graphic parameters also showed varied distribution.

The E value varied between 0.6 to 1.10 m/s.

The A value varies between 0.55 to 1.10 m/s.

E’ value varied between 4.2 to 9.6 cm/s. (mean =6.8)

E/e” value ranged between 7.5 to 20, with a mean of 12.62

TABLE .10 -KILLIP CLASS

| KILLIP CLASS | N | % |
|---|---|---|
| I | 20 | 40.0 |
| II | 24 | 48.0 |
| III | 5 | 10.0 |
| IV | 1 | 2.0 |
| Total | 50 | 100.0 |

## KILLIP CLASS AND PRESENTATION

Of the total of 50 patients 44 patients presented with killip class I-II

The remaining 6 patients presented with killip class III-IV

The killip class I-II group comprised 88% of the total patients and killipclass III-IV group comprised 12% of the total admissions.

TABLE .11 - DEATH

| DEATH | N | % |
|---|---|---|
| Yes | 7 | 14.0 |
| No | 43 | 86.0 |
| Total | 50 | 100 |

**MORTALITY RATE**

The mortality rate in the overall study group was 14% with

7 of the total 50 patients admitted had in-hospital deathThis rate was slightly higher than the routine mortality rate of about 7- 8% in our coronary care unit.

TABLE .12- DEATH AND EJECTION FRACTION

| | | | Death | | Total | P value |
|---|---|---|---|---|---|---|
| | | | Yes | No | | |
| EF | < 45 | Count | 6 | 19 | 25 | 0.042* |
| | | % within EF | 24.0% | 76.0% | 100.0% | |
| | | % within Death | 85.7% | 44.2% | 50.0% | |
| | > 45 | Count | 1 | 24 | 25 | |
| | | % within EF | 4.0% | 96.0% | 100.0% | |
| | | % within Death | 14.3% | 55.8% | 50.0% | |
| Total | | Count | 7 | 43 | 50 | |

Left ventricular systolic function is very good prognostic indicator in predicting in hospital mortality in patients with acute myocardial infraction.

In our study patients with an EF of < 45% had a mortality rate of 24% as against a mortality rate of only 4% for patients with an EF of > 45%.

This was is consonance with the results of major studies in myocardial infraction which higher incidence of heart failure, ventricular arrhythmias, and both short-term and long-term mortality rates.

FIGURE .7 -   DEATH AND EJECTION FRACTION

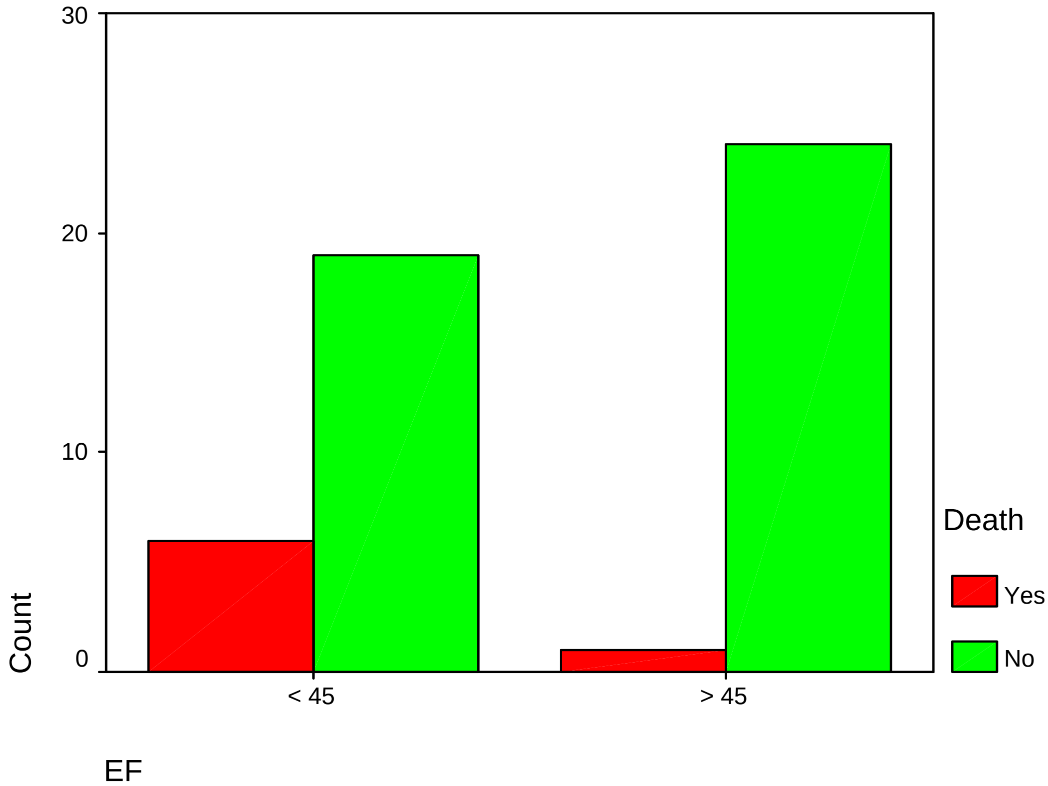


TABLE .13 -DT AND MORTALITY

| | | | Death | | P value |
|---|---|---|---|---|---|
| | | | Yes | No | |
| DT (ms ) | < 140 | Count | 6 | 10 | |
| | | % within DT (ms ) | 37.5% | 62.5% | |
| | | % within Death | 85.7% | 23.3% | |
| | > 140 | Count | 1 | 33 | |
| | | % within DT (ms ) | 2.9% | 97.1% | 0.001 |
| | | % within Death | 14.3% | 76.7% | |
| Total | | Count | 7 | 43 | |
| | | % within DT (ms ) | 14.0% | 86.0% | |
| | | % within Death | 100.0% | 100.0% | |

**DT and Mortality**

The mitral valve deceleration time (DT) is an indicator of rapid filling phase and hence the left ventricular . With decreasing LV compliance the DT is shortened progressively to < 140 ms

In the present study, a DT of <140 ms correlated with increased mortality rates at 37.5% and the mortality rate was only 2.9% when the DT was > 140 ms

In the study of Hillisetal, DT did not correlate well with mortality where it was only E/e' ratio which had a better correlation with mortality.

In our study a DT < 140 ms was also an independent and adverse predictor of mortality during the in- hospital stay of the patient.

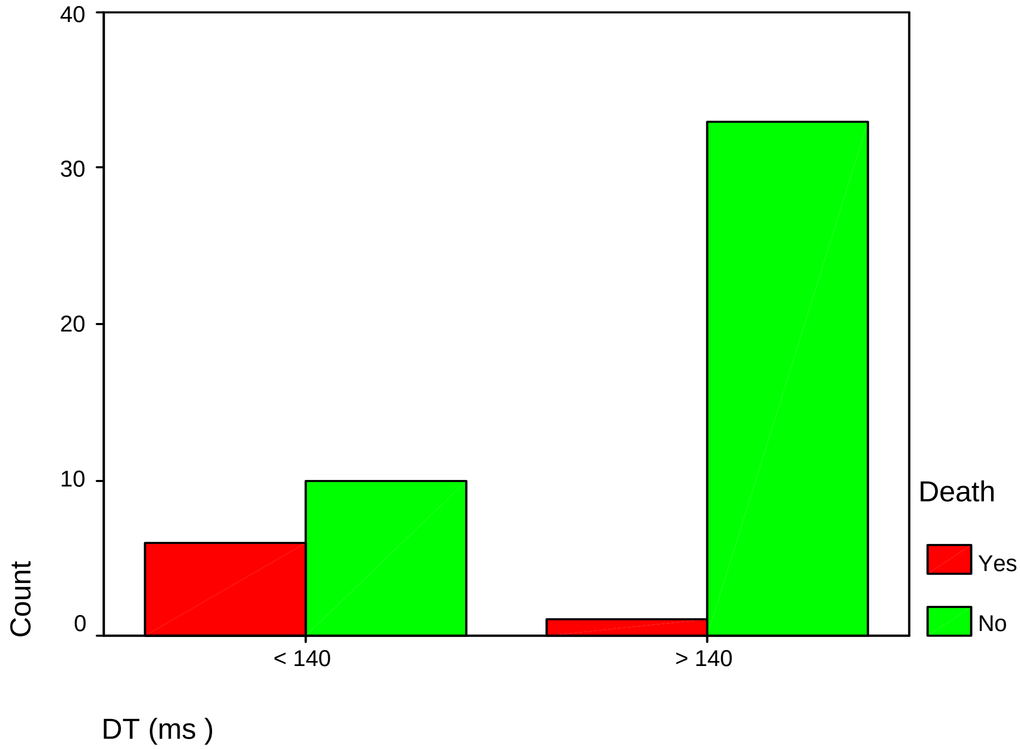
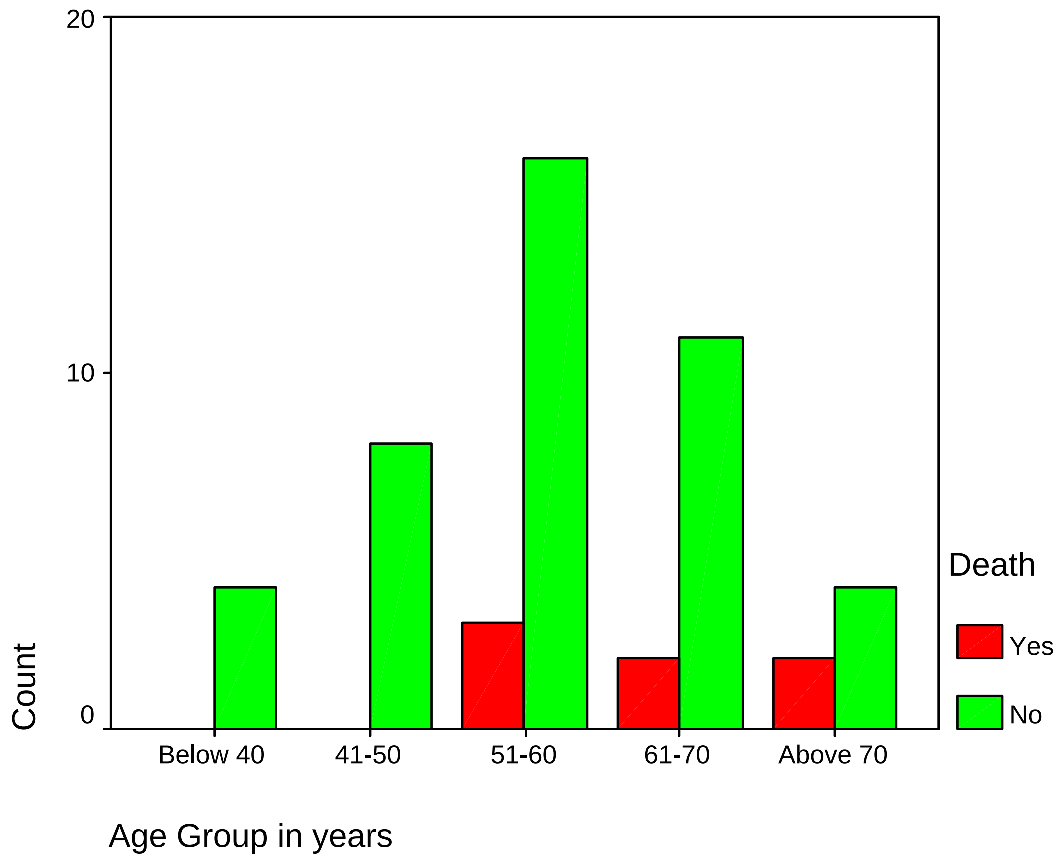



FIGURE .8 – DT AND MORTALITY

FIGURE .9 - AGE- WISE MORTALITY




**AGE WISE MORTALITY**

Of the different age group of patients admitted with acute coronary syndrome, the mortality was highest in the age group of 70 years and above (50%) and no deaths in the age group below 50 years of age.

All the 7 deaths that occurred happened in the age group 50 years and above, with two deaths in the age group of 70 years and above.

TABLE .14 – KILLIP CLASS AND MORTALITY

| | | | Death | | P Value |
|---|---|---|---|---|---|
| | | | Yes | No | |
| KILLIP | I | Count | 1 | 19 | |
| | | % within KILLIP | 5.0% | 95.0% | |
| | | % within Death | 14.3% | 44.2% | |
| | II | Count | 3 | 21 | |
| | | % within KILLIP | 12.5% | 87.5% | |
| | | % within Death | 42.9% | 48.8% | |
| | III | Count | 3 | 2 | |
| | | % within KILLIP | 60.0% | 40.0% | |
| | | % within Death | 42.9% | 4.7% | .04 |
| | IV | Count | 0 | 1 | |
| | | % within KILLIP | .0% | 100.0% | |
| | | % within Death | .0% | 2.3% | |
| Total | | Count | 7 | 43 | |
| | | % within KILLIP | 14.0% | 86.0% | |
| | | % within Death | 100.0% | 100.0% | |

**KILLIP CLASS AND MORTALITY**

The killip class devised in the pre –thrombolytic era to predict mortality in patients with coronary syndromes still holds good even in this era of catheter based reperfusion.

The mortality was highest in the killip II-III group accounting for 85% of the total deaths. Since there was only one patient admitted with killip class IV who was promptly treated with a percutaneous coronary intervention and device closure of ventricular septal rupture that patient survived to hospital discharge.

FIGURE .10 – KILLIP CLASS AND MORTALITY

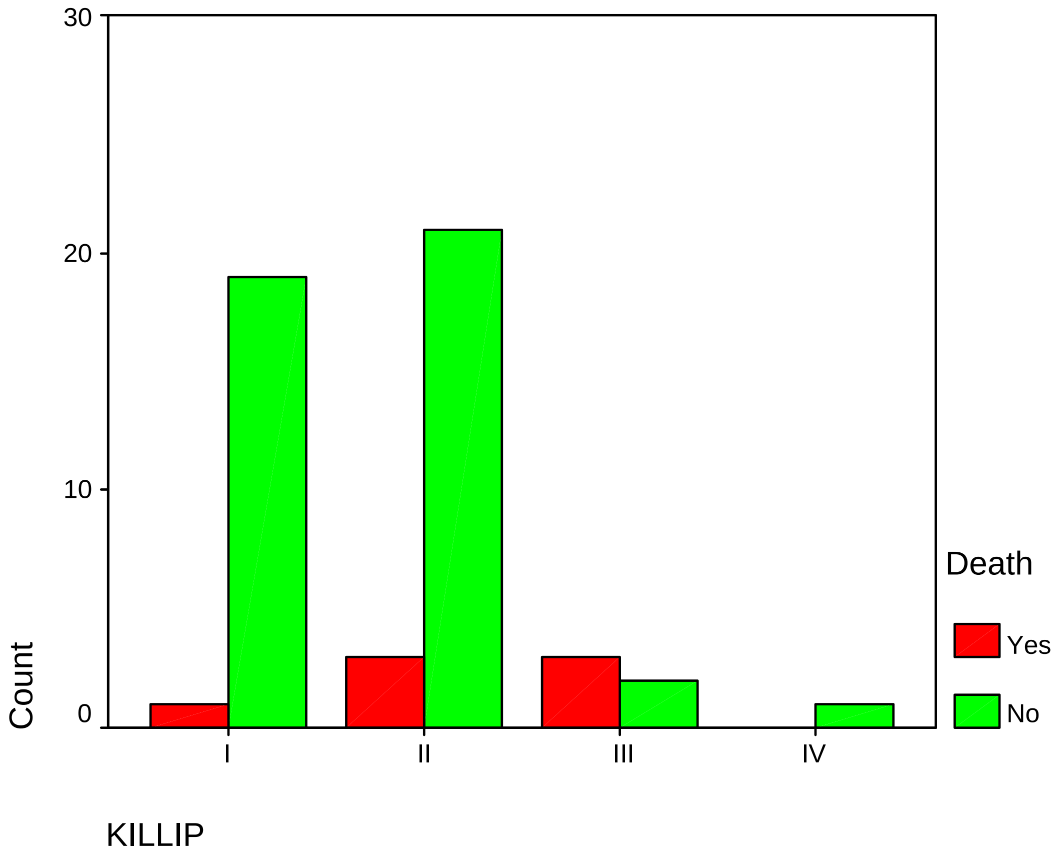


TABLE .17 – EF AND DECELERATION TIME

| | | | EF | | Total |
|---|---|---|---|---|---|
| | | | < 45 | > 45 | |
| DT (ms ) | < 140 | Count | 15 | 1 | 16 |
| | | % within DT (ms ) | 93.8% | 6.3% | 100.0% |
| | | % within EF | 60.0% | 4.0% | 32.0% |
| | > 140 | Count | 10 | 24 | 34 |
| | | % within DT (ms ) | 29.4% | 70.6% | 100.0% |
| | | % within EF | 40.0% | 96.0% | 68.0% |
| Total | | Count | 25 | 25 | 50 |
| | | % within DT (ms ) | 50.0% | 50.0% | 100.0% |
| | | % within EF | 100.0% | 100.0% | 100.0% |

DT was generally shortened in patients with ejection fraction of< 45%. More patients had on DT < 140 ms (n=15) if the ejection fraction was <45% than when it was >45% (n=1). On the other hand if the ejection fraction was above 45% the DT was maintained above 140 ms only 1 patient with ejection fraction of > 45% had on DT of < 140 ms.

Hence normal DT had a higher positive predictive value to predict a higher ejection fraction > 45%.

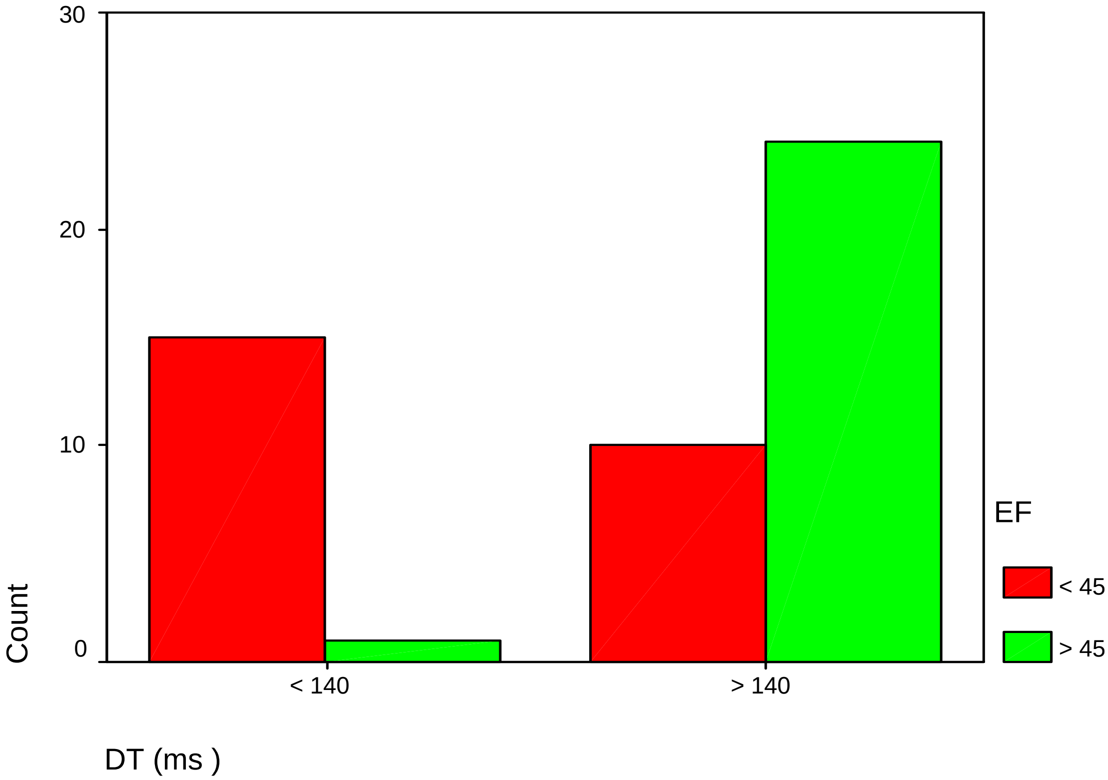



FIGURE .11– EF AND DECELERATION TIME

**E/e’ RATIO ,ITS DISTRIBUTION AND CORRELATION.**

TABLE .18 – DISTRIBUTION OF E/e’

| **E/e’** | |
|---|---|
| <15 | >15 |
| 34 | 16 |
| 68% | 32% |

34 of the total 50 patients had an E /e’ ratio of <15 and the remaining 16 patients had an E/e’ of >15. This constituted 68% and 32% of the total study participants respectively.

A majority of patients (68%) in the study group had on E/e of < 15 indicating normal LV filling pressures.

Among the patients with elevated filling pressures as manifested by an E/e’ ration of>15 nearly 88% of the patients had an abbreviated DT at < 140 ms.

Whereas in the group with normal E/e’ ratio of < 15, only 6% had an shortened DT at < 140 ms .Both these findings suggest that there is a strong correlation between an elevated E/e’ and shortened DT.

TABLE.20 - E/e' AND DT

| **DT** | **E/e'** | |
|---|---|---|
| | <15 | >15 |
| >140 | 32 | 2 |
| <140 | 2 | 14 |

TABLE .21 - E/e' AND DEATH

| **P Value 0.001** | | **E/e'** | |
|---|---|---|---|
| | | >15 | <15 |
| DEATHS | N | 6/16 | 1/34 |
| | % | 37.5 | 2.94 |

TABLE.22 – E/e’  AND EJECTION  FRACTION

| **EF** | **E/e’** | |
|---|---|---|
| | **<15** | **>15** |
| <45 | 9 | 16 |
| >45 | 25 | 0 |

## E/e’ AND EJECTION FRACTION

None of the patients with an ejection fraction of >45 had an elevated E/e’ ratio. But in the patient group with a reduced ejection fraction. The E/e’ ratio was elevated at >15 in 64% of the total of 25 patients and was normal in the remaining 36% of the patients with reduced ejection fraction  group.

## E/e’ AND MORTALITY

E/e’ had a strong correlation in predicting death as an outcome of the total of 16 patients with an E/e’ ratio of > 15 suggestive of elevated left ventricular filling pressures .6 of these patients did not survive to hospital discharge amounting to a mortality rate of **37.5%** in that group ( Average mortality 14% )

In contrast in patients who had an E/e’ ratio of < 15 the mortality rate was low at **2.94%.**

Thus E/e’ was an independent predictor of hospital mortality in all patients admitted with acute myocardial infraction irrespective of baseline characters, risk factors and ejection fraction of the patient.

# CONCLUSIONS

Left ventricular systolic function has been a conventional and established risk stratification tool ever since echocardiography came to be used routinely in the setting of acute myocardial infarction (30).The present study also reliably highlights the fact that patients with lower ejection fraction had a higher in hospital mortality compared with patients with higher or normal ejection fraction (31).

The present study used the Doppler parameters of diastolic function of the left ventricle as a prognostic tool in prediction of in-hospital events in patients with acute myocardial infraction.

The Doppler parameters studied were E velocity, A velocity ,E/A ratio, e' tissue Doppler velocity, E wave deceleration Time and E/e' ratio.

## E velocity, A velocity, E/A Ratio

E velocity, A velocity and E/A ration in isolation did not reliaby predict the outcomes in patients admitted in acute coronary syndromes. They showed wide variations among patients with similar ejection fraction and in-hospital outcomes.

However the other indices of diastolic function like the DT(Deceleration Time), e' tissue Doppler velocity of lateral mitral annulus and E/e' ratio reliably predicted in-hospital outcomes in patients with ST elevation myocardial infraction.

**<u>Deceleration Time (DT)</u>**

In contra distinction to previous studies (19). The present study showed a good correlation between a reduced DT and negative patient outcomes. 6 out of the 7 patients who died during their in-hospital stay had a DT of <140ms

Similarly in the overall group of patients who had an abbreviated DT<140ms,

the morality rates were higher (37.5)% than in the group which had a normal DT (> 140ms) (2.9%).

A reduced DT hence predicted an elevated LV filling pressure, a large area of infarct and adverse in-hospital mortality trends in patients admitted with acute myocardial infraction.

**<u>e’ velocity:</u>**

The tissue Doppler e’ velocity of the lateral mitral annulus also reliably predicted outcomes in patients during their in-hospital stay.

In general patients who has an e’ velocity of >6 cm/s had relatively lesser mortality rates than in patients who had an e’ velocity of < 6cm/s

But this factor did not correlate well with ejection fraction or Deceleration time.

**<u>E/e’ ratio:</u>**

Among the three Doppler indices which reliablypredicted the in-hospital outcomes in patients, E/e’ ratio was the most consistent. E/e’ reliably predicted in –hospital mortality, correlated better with the patient’s ejection fraction and also with the mitral valve deceleration time (DT).

Patients with higher E/e’ ratio (>15) had an higher hospital mortality (37.5%).

The hospital mortality rate was low at 3% in the patient groups with low E/e’ ratios (<15)

In addition, 6 out of 7 patients who dies in the hospital had an increased E/e’ ratio (>15) and only one patient had a reduced E/e’ ratio.

The E/e’ ration also reliably predicted the ejection fraction.

None of the patients with a EF of > 45% had an E/e’ ratio of >15

While in the group with an EF of < 45% nearly 64% of patients had an E/e’ of >15.

This indirectly predicted that patients with elevates E/e’ (>15) had a lower ejection fraction, elevated LV filling pressures as a result of larger area of left ventricular myocardium that has been damaged.

This is turn predicted poorer patients outcomes with greatly increased in-hospital mortality rates.

Thus E/e’ ratio can be used as a surrogate marker of elevated LV filling pressure and hence can be reliably used as a prognostic marker to risk strictly presents admitted in coronary care units over and above other non-invasive tools already available.

## LIMITATIONS OF THE STUDY

i) Smaller sample size (N=50) and hence the results of the study cannot be extrapolated to general patient groups.

ii) Shorter follow-up of patients during their hospital stay only and the need for long term follow up to predict outcomes

iii) Most patients did not undergo invasive studies [coronary angiograms and measurement of LV filling pressures (PCWP) ].

iv) Only a single Echocardiographic evaluation within 24 hours of admission was done and no serial follow-up studies undertaken.

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| AGE | SEX | SMOKER | T2DM | HTN | DYSLIPID. | PRE. MI | PREV.REVA | KILLIP |
|---|---|---|---|---|---|---|---|---|
| 60 | M | N | Y | Y | Y | N | N | II |
| 45 | M | Y | N | N | Y | N | N | II |
| 56 | M | Y | N | Y | Y | N | N | I |
| 68 | F | N | Y | Y | Y | N | N | II |
| 39 | F | N | Y | Y | Y | N | N | II |
| 41 | M | Y | Y | N | Y | N | N | II |
| 42 | M | Y | N | N | Y | N | N | I |
| 65 | M | Y | N | Y | Y | N | N | II |
| 42 | F | N | Y | N | N | N | N | I |
| 52 | M | Y | Y | N | N | Y | N | II |
| 67 | M | Y | Y | N | Y | N | N | III |
| 55 | M | Y | Y | N | N | N | N | III |
| 60 | M | Y | N | N | N | N | N | II |
| 55 | M | Y | N | N | Y | N | N | I |
| 75 | M | N | Y | N | Y | N | N | I |
| 42 | M | Y | Y | N | N | N | N | II |
| 51 | M | Y | N | Y | N | N | N | I |
| 55 | M | Y | N | N | N | N | N | I |
| 66 | M | N | Y | Y | Y | N | N | I |
| 54 | M | Y | N | N | N | N | N | II |
| 51 | M | N | N | Y | Y | N | N | I |
| 61 | M | Y | N | Y | N | N | N | II |
| 55 | M | Y | N | Y | Y | N | N | II |
| 55 | F | N | Y | Y | Y | N | N | II |
| 72 | F | N | Y | Y | N | N | N | II |
| 61 | M | Y | N | Y | N | N | N | I |
| 68 | M | N | Y | N | Y | N | N | III |
| 65 | F | N | Y | Y | N | N | N | II |
| 70 | F | N | Y | N | Y | N | N | III |
| 55 | M | Y | Y | N | N | N | N | II |
| 60 | M | Y | Y | N | Y | N | N | I |
| 90 | F | N | Y | Y | N | N | N | II |
| 29 | M | Y | N | N | N | N | N | I |
| 40 | M | Y | N | N | N | N | N | I |
| 57 | M | Y | N | Y | N | N | N | II |
| 67 | M | Y | N | Y | N | N | N | IV |
| 70 | M | N | Y | Y | N | N | N | I |
| 55 | M | N | N | Y | Y | N | N | I |
| 48 | M | Y | N | N | Y | N | N | II |
| 46 | M | N | N | Y | Y | N | N | I |
| 61 | M | Y | N | Y | Y | N | N | II |
| 65 | F | N | Y | Y | N | N | N | II |
| 45 | M | N | N | Y | N | N | N | I |
| 72 | F | N | Y | N | N | N | N | II |
| 53 | M | Y | N | N | Y | N | N | I |
| 75 | M | N | Y | N | Y | N | N | III |
| 72 | M | N | Y | Y | N | N | N | II |
| 60 | M | Y | N | Y | Y | N | N | II |
| 35 | M | Y | N | N | N | N | N | I |
| 52 | M | N | Y | N | N | N | N | I |

| TW (HRS) | ANT.MI | INF.MI | SK | PCI/CABG | IONO | I.V NTG | CS | DEATH |
|---|---|---|---|---|---|---|---|---|
| 3 | Y | N | Y | N | Y | Y | N | N |
| 18 | Y | N | N | N | N | Y | N | N |
| 5 | Y | N | Y | N | N | Y | N | N |
| 9 | N | Y | Y | N | Y | N | N | N |
| 7 | N | Y | Y | N | N | N | N | N |
| 7 | Y | N | Y | N | N | Y | N | N |
| 3.5 | N | Y | Y | N | N | N | N | N |
| 3 | Y | N | Y | N | N | Y | N | N |
| 5 | Y | N | Y | N | N | N | N | N |
| 6 | N | Y | Y | N | N | N | N | N |
| 9 | Y | N | Y | N | Y | Y | Y | Y |
| 6 | Y | N | Y | N | Y | Y | N | N |
| 6 | N | Y | Y | N | Y | Y | N | N |
| 7 | N | Y | Y | N | Y | N | N | N |
| 16 | N | Y | N | N | N | Y | N | N |
| 20 | Y | N | N | N | Y | Y | N | N |
| 18 | N | Y | N | N | N | Y | N | N |
| 7 | N | Y | Y | N | N | N | N | N |
| 2 | Y | N | Y | N | N | Y | N | N |
| 16 | Y | N | N | N | N | Y | N | N |
| 3.5 | N | Y | Y | N | N | N | N | N |
| 8 | Y | N | Y | N | N | Y | N | N |
| 7 | N | Y | Y | N | Y | N | N | Y |
| 8.5 | Y | N | Y | N | N | Y | N | N |
| 16 | N | Y | N | N | Y | N | N | N |
| 4 | Y | N | Y | N | N | N | N | N |
| 9 | Y | N | Y | N | Y | Y | N | N |
| 6.5 | Y | N | Y | N | N | N | N | N |
| 9 | N | Y | Y | N | Y | N | Y | Y |
| 11 | Y | N | Y | N | Y | N | Y | Y |
| 7 | Y | N | Y | N | N | N | N | N |
| 9 | Y | N | N | N | N | N | N | N |
| 5 | Y | N | Y | N | N | N | N | N |
| 9 | Y | N | Y | N | N | N | N | N |
| 16 | N | Y | N | N | N | N | N | N |
| 8 | Y | N | Y | Y | Y | Y | Y | N |
| 6 | Y | N | Y | N | N | N | N | N |
| 8 | Y | N | Y | N | N | N | N | N |
| 6 | Y | N | Y | N | Y | N | N | N |
| 2 | Y | N | Y | N | N | N | N | N |
| 7 | Y | N | Y | N | N | Y | N | N |
| 9 | N | Y | Y | N | N | Y | N | N |
| 3 | Y | N | Y | N | N | N | N | N |
| 11 | N | Y | Y | N | N | N | N | N |
| 3 | N | Y | Y | N | N | N | N | N |
| 12 | Y | N | Y | N | Y | N | N | Y |
| 6 | N | Y | Y | N | Y | N | Y | Y |
| 8 | Y | N | Y | N | N | Y | N | N |
| 18 | N | Y | N | N | N | N | N | N |
| 7 | N | Y | Y | N | N | N | N | Y |

| EF | LVEDD(mm | LVESD | E(m/s) | A (m/s) | DT (ms ) | e'( cm/s) | E/e' | | MR |
|---|---|---|---|---|---|---|---|---|---|
| >45 | 44 | 30 | 0.72 | 0.66 | >140 | 8.4 | | 8.5 | N |
| <45 | 52 | 42 | 0.86 | 0.9 | >140 | 7.2 | | 12 | N |
| >45 | 46 | 31 | 0.78 | 0.82 | >140 | 7 | | 11.1 | N |
| >45 | 46 | 32 | 0.75 | 0.8 | >140 | 8 | | 9.3 | N |
| <45 | 52 | 42 | 0.72 | 0.8 | >140 | 5.8 | | 12.4 | N |
| <45 | 51 | 40 | 0.8 | 0.9 | >140 | 6.1 | | 12 | N |
| <45 | 44 | 29 | 0.8 | 0.72 | >140 | 9.6 | | 8 | N |
| <45 | 48 | 32 | 0.9 | 0.7 | <140 | 5.62 | | 16 | N |
| >45 | 49 | 31 | 0.7 | 0.6 | >140 | 7.2 | | 9.7 | N |
| >45 | 48 | 33 | 0.75 | 0.6 | >140 | 6.5 | | 11.5 | N |
| <45 | 52 | 42 | 0.72 | 0.82 | <140 | 4.5 | | 16 | N |
| <45 | 51 | 40 | 0.8 | 0.55 | <140 | 5.1 | | 16 | N |
| >45 | 47 | 32 | 0.76 | 0.8 | >140 | 6.3 | | 12 | N |
| >45 | 45 | 31 | 0.8 | 0.72 | >140 | 9.2 | | 8.7 | N |
| >45 | 44 | 30 | 0.9 | 0.7 | <140 | 6.2 | | 14.5 | N |
| <45 | 51 | 42 | 0.76 | 0.8 | <140 | 4.8 | | 15.8 | N |
| >45 | 43 | 29 | 0.6 | 0.55 | >140 | 8 | | 7.5 | N |
| >45 | 44 | 28 | 0.77 | 0.6 | >140 | 9.6 | | 8 | N |
| >45 | 44 | 30 | 0.82 | 0.7 | >140 | 9.2 | | 8.9 | N |
| <45 | 48 | 38 | 0.8 | 0.9 | >140 | 9 | | 8 | N |
| >45 | 44 | 31 | 0.8 | 0.7 | >140 | 8.2 | | 9.8 | N |
| <45 | 48 | 37 | 0.76 | 0.82 | >140 | 4.5 | | 16.8 | N |
| <45 | 46 | 35 | 0.7 | 0.8 | <140 | 4.2 | | 16.6 | N |
| <45 | 48 | 36 | 0.72 | 0.75 | >140 | 5 | | 14.4 | N |
| <45 | 52 | 42 | 0.75 | 0.6 | <140 | 4.6 | | 16.3 | N |
| >45 | 45 | 32 | 0.8 | 0.7 | >140 | 9 | | 8.1 | N |
| <45 | 49 | 38 | 1 | 0.7 | <140 | 5 | | 20 | N |
| <45 | 50 | 40 | 0.9 | 0.7 | <140 | 5 | | 18 | N |
| <45 | 48 | 36 | 0.9 | 0.7 | <140 | 4.8 | | 18.8 | N |
| <45 | 52 | 43 | 0.9 | 0.7 | <140 | 5 | | 18 | Y |
| >45 | 48 | 32 | 0.7 | 0.6 | >140 | 7 | | 10 | N |
| <45 | 53 | 42 | 1 | 1.1 | <140 | 6 | | 16.6 | N |
| >45 | 45 | 30 | 0.7 | 0.6 | >140 | 8 | | 8.8 | N |
| >45 | 48 | 30 | 0.8 | 0.7 | >140 | 6 | | 13 | N |
| <45 | 52 | 42 | 0.9 | 0.8 | >140 | 8 | | 11.2 | N |
| <45 | 52 | 40 | 0.9 | 0.8 | <140 | 8 | | 11.2 | N |
| >45 | 45 | 32 | 0.8 | 0.6 | >140 | 7 | | 11 | N |
| <45 | 49 | 38 | 0.6 | 0.7 | >140 | 6 | | 11.6 | N |
| <45 | 55 | 46 | 1 | 0.7 | <140 | 5.1 | | 20 | Y |
| >45 | 45 | 30 | 0.7 | 0.6 | >140 | 8 | | 8.1 | N |
| >45 | 50 | 35 | 0.7 | 0.8 | >140 | 7 | | 10 | N |
| >45 | 45 | 31 | 0.8 | 0.7 | >140 | 9 | | 9 | N |
| >45 | 45 | 32 | 0.9 | 0.7 | >140 | 9.2 | | 9.8 | N |
| >45 | 48 | 32 | 0.7 | 0.8 | >140 | 7 | | 10 | N |
| >45 | 45 | 32 | 0.9 | 0.8 | >140 | 8.2 | | 11 | N |
| <45 | 52 | 40 | 1 | 0.7 | <140 | 5.5 | | 18 | N |
| <45 | 48 | 39 | 1.1 | 0.8 | <140 | 5.5 | | 20 | N |
| <45 | 50 | 41 | 0.7 | 0.8 | >140 | 4.5 | | 15.5 | N |
| >45 | 46 | 32 | 0.9 | 0.6 | >140 | 8.1 | | 11 | N |
| >45 | 48 | 32 | 1 | 0.8 | >140 | 8 | | 12.5 | N |

| E/A |
|---|
| 1.1 |
| 0.95 |
| 0.95 |
| 0.93 |
| 0.9 |
| 0.88 |
| 1.1 |
| 1.3 |
| 1.2 |
| 1.25 |
| 0.87 |
| 1.45 |
| 0.95 |
| 1.1 |
| 1.3 |
| 0.9 |
| 1.1 |
| 1.3 |
| 1.3 |
| 0.88 |
| 1.3 |
| 0.9 |
| 0.88 |
| 0.96 |
| 1.25 |
| 1.3 |
| 1.42 |
| 1.3 |
| 1.3 |
| 1.3 |
| 1.2 |
| 0.9 |
| 1.12 |
| 1.3 |
| 1.12 |
| 1.12 |
| 1.33 |
| 0.85 |
| 1.42 |
| 1.2 |
| 0.9 |
| 1.3 |
| 1.3 |
| 0.9 |
| 1.3 |
| 1.43 |
| 1.4 |
| 0.8 |
| 1.5 |
| 1.25 |

# ECHOCARDIOGRAPHIC ESTIMATION OF LEFT VENTRICULAR FILLING PRESSURE BY E/e’ AS A PREDICTOR OF SURVIVAL AFTER ACUTE MYOCARDIAL INFARCTION

## PROFORMA

Name : Age : Sex :

IP No. / CD No. :

**RISK FACTORS**

| | | |
|---|---|---|
| 1. | Male Gender | |
| 2. | Smoker | |
| 3. | Diabetes Mellitus | |
| 4. | Hypertension | |
| 5. | Hyperlipidemia | |
| 6. | Previous MI | |
| 7. | Previous Revascularisation | |

**CLINICAL CHARACTERISTICS**

| | | |
|---|---|---|
| 1. | Killip Class | |
| 2. | Median Delay | |
| 3. | Anterior MI | |
| 4. | Inferior MI | |
| 5. | Thrombolytic Therapy | |

**IN-HOSPITAL REVASCULARISATION**

| | | | |
|---|---|---|---|
| 1. | Thrombolysis | Successful | |
| | | Failed | |
| 2. | PPCI / RESCUE PCI | | |
| 3. | DELAYED PCI | | |
| 4. | CABG | | |

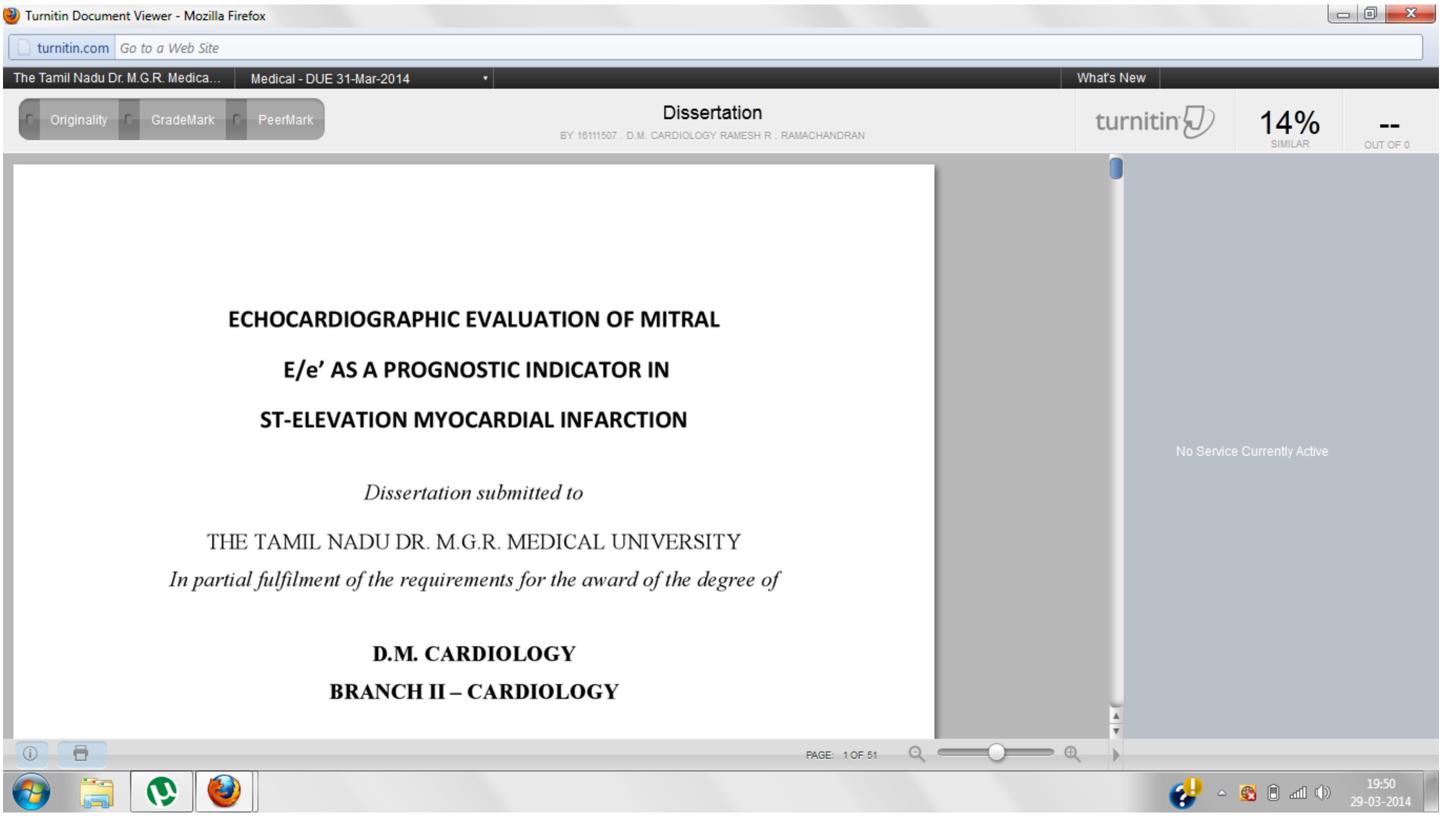
| | |
|---|---|
| **MULTILEVEL CORONARY DISEASE** | |

## IN-HOSPITAL THERAPY & EVENTS

| | | |
|---|---|---|
| 1. | HEPARIN | |
| 2. | I.V. NITROGLYCERIN | |
| 3. | IONOTROPIC SUPPORT | |
| 4. | ELECTRICAL COMPLICATIONS | |
| 5. | MECHANICAL COMPLICATIONS | |
| 6. | CARDIOGENIC SHOCK | |
| 7. | DEATH | |

## ECHOCARDIOGRAPHIC CHARACTERISTICS

| S.NO. | TIMING OF ECHOCARDIOGRAPHY | | ON ADMISSION | 24 HOURS |
|---|---|---|---|---|
| 1. | LVEF (%) | >45% | | |
| | | <45% | | |
| 2. | LVEDD (mm) | | | |
| 3. | LVESD (mm) | | | |
| 4. | Peak E-Wave velocity (m/s) | | | |
| 5. | Peak A-Wave velocity (m/s) | | | |
| 6. | E/A Ratio | | | |
| 7. | DT (ms) | < 140 | | |
| | | > 140 | | |
| 8. | Mitral annulus e’ cm/s | | | |
| 9. | E/e’ | | | |
| 10. | Moderate / Severe MR | | | |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ST-ELEVATION MYOCARDIAL INFARCTION
Dissertation submitted to
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
In partial fulfilment of the requirements for the award of the degree of
D.M. CARDIOLOGY
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29-03-2014